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United States Court of Appeals
Tenth Circuit

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PATRICK FISHER
Clerk

PUBLISH

UNITED STATES COURT OF APPEALS

TENTH CIRCUIT

RICHARD W. GOEBEL, Plaintiff - Appellee, vs. THE DENVER AND RIO GRANDE WESTERN RAILROAD COMPANY, a Delaware corporation, Defendant - Appellant.	No. 02-1391

APPEAL FROM THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF COLORADO
(D.C. No. 94-N-2206 (BNB))

Lawrence M. Mann, Alper & Mann, P.C., Washington, D.C., (and Christopher B. Little, Montgomery, Little & McGrew, P.C., Englewood, Colorado, with him on the brief), for Plaintiff - Appellee.

James W. Erwin (and Thomas R. Jayne, Thompson, Coburn, L.L.P., St. Louis, Missouri, and Steven E. Napper, Denver, Colorado, on the briefs), for Defendant - Appellant.

Before **KELLY**, **HENRY**, and **HARTZ**, Circuit Judges.

KELLY, Circuit Judge.

In June 1999, Plaintiff-Appellee Richard W. Goebel obtained a $755,000 jury verdict against his employer, Defendant-Appellant The Denver and Rio Grande Western Railroad Company ("Railroad"), for injuries suffered on the job. The Railroad appealed and we concluded that the district court failed to perform its gatekeeper function and thus abused its discretion by admitting the testimony of plaintiff expert Dr. Daniel Teitelbaum. Goebel v. Denver and Rio Grande W. R.R. Co., 215 F.3d 1083, 1088 (10th Cir. 2000) ("Goebel I"). Accordingly, we reversed and remanded for a new trial. Id. at 1089.

On remand, the Railroad renewed its motion to exclude Dr. Teitelbaum's testimony under Daubert v. Merrell Dow Pharms., Inc., 509 U.S. 579 (1993). After denying the Railroad's request for an evidentiary hearing, the district court denied the Railroad's Daubert motion in a thorough written order. To expedite appeal on the core issue, the parties stipulated to a judgment in Mr. Goebel's favor in an amount equal to the initial jury award, with the Railroad reserving its right to appeal the district court's Daubert decision. After the court entered judgment, the Railroad filed this appeal arguing that the district court abused its discretion in admitting Dr. Teitelbaum's testimony. We exercise jurisdiction pursuant to 28 U.S.C. § 1291 and affirm.

Background

Mr. Goebel claims that in January 1994, he was injured on the job during a mishap in the Moffat Tunnel in Colorado which involved exposure to high elevations and diesel fumes. Details of the mishap as described in Goebel I are incorporated by reference. 215 F.3d at 1085-87. As we noted then, Mr. Goebel sued the Railroad under the Federal Employers' Liability Act alleging personal injuries resulting from the tunnel incident. The district court granted summary judgment to Mr. Goebel on the question of liability and limited the trial to issues of causation and damages. At trial, Dr. Teitelbaum testified as to the causation of Mr. Goebel's injuries:

> I believe that the cause of Mr. Goebel's injury was his exposure to a unique environment, deficient in oxygen at low barometric pressure, contaminated with pulmonary irritants, which combined with the unique physiologic setting which takes place at high altitude produced an oxygen lack syndrome, which produced swelling in his brain, called cerebral edema, which resulted in small diffuse pressure injuries which resulted in his cognitive defect.
>
> It's a complicated chain of events, but one which is relatively simple to explain on the basis of the fundamental physiology. All of these pieces have been looked at in separate events. In this gentleman, they occurred at the same time and produced this result.

I R. at 50. The jury found in favor of Mr. Goebel and awarded him $755,000 in damages.

Discussion

I. Standard of Review

As we stated in Goebel I, we review de novo the question of whether the district court performed its gatekeeper role and applied the proper legal standard in admitting an expert's testimony. 215 F.3d at 1087; see also Dodge v. Cotter Corp., 328 F.3d 1212, 1223 (10th Cir. 2003). We then review for abuse of discretion the trial court's actual application of the gatekeeper standard in deciding whether to admit or exclude an expert's testimony. General Elec. Co. v. Joiner, 522 U.S. 136, 142 (1997). The trial court's broad discretion applies both in deciding how to assess an expert's reliability, including what procedures to utilize in making that assessment, as well as in making the ultimate determination of reliability. Kumho Tire Co. v. Carmichael, 526 U.S. 137, 152 (1999); Dodge, 328 F.3d at 1223. Accordingly, we will not disturb the district court's ruling unless it is "arbitrary, capricious, whimsical or manifestly unreasonable" or when we are convinced that the district court "made a clear error of judgment or exceeded the bounds of permissible choice in the circumstances." Dodge, 328 F.3d at 1223 (quoting Atlantic Richfield Co. v. Farm Credit Bank of Wichita, 226 F.3d 1138, 1163-64 (10th Cir. 2000)).

Neither party argues that the district court has again failed to perform its gatekeeper function. In fact, as we required on remand, when faced with the

Railroad's renewed Daubert objection, the district court took pains to "demonstrate by specific findings on the record that it ha[d] performed its duty as gatekeeper." Goebel I, 215 F.3d at 1088.

Furthermore, although the district court denied the Railroad's request for a Daubert hearing, III R. at 661, the Railroad has not appealed that ruling. As a result, our task here is simply to review the district court's detailed findings in light of the record and to apply the deferential abuse of discretion standard to its decision to admit Dr. Teitelbaum's testimony. The Railroad's decision not to appeal the denial of an evidentiary hearing prompts us to note that our review is appropriately constrained by the record developed by the parties. In cases such as this one, where one party alleges that an expert's conclusions do not follow from a given data set, the responsibility ultimately falls on that challenging party to inform (via the record) those of us who are not experts on the subject with an understanding of precisely how and why the expert's conclusions fail to follow from the data set. Any failure by the challenging party to satisfy this responsibility is at that party's peril.

II. An Overview of the Railroad's Arguments

Because it provides a convenient means of analyzing the district court's ruling, we believe Dr. Teitelbaum's opinion is best viewed as a conclusion that two separate aspects of causation existed in this case: (1) general causation,

meaning that the particular circumstances in the tunnel *could* have caused Mr. Goebel's injury, and (2) specific causation, meaning that those circumstances *did in fact* cause Mr. Goebel's injury. See, e.g., Soldo v. Sandoz Pharms. Corp., 244 F.Supp.2d 434, 524-25 (W.D. Pa. 2003) (discussing these concepts and collecting cases). To arrive at general causation, Dr. Teitelbaum reviewed scientific literature, drew general propositions therefrom, and then combined those propositions to conclude that the conditions in the tunnel *could* have caused high altitude cerebral edema ("HACE") in Mr. Goebel. III R. at 664-65. To arrive at specific causation, Dr. Teitelbaum performed a differential diagnosis in which, after examining and testing Mr. Goebel, he ruled in all scientifically plausible causes of the injury and then ruled out the least plausible causes until only the most likely cause remained, i.e., that the tunnel conditions *did in fact* cause Mr. Goeble to suffer HACE-induced cognitive deficits. Id. at 668-70.

The Railroad contends that the district court abused its discretion by admitting Dr. Teitelbaum's testimony because (1) his general causation opinion was not supported by the medical literature he relied upon and (2) his differential diagnosis was unreliable because he failed to account for alternative explanations of Mr. Goebel's condition. Aplt. Br. at 15-16. Because the Railroad's arguments on appeal are the same as those made before the district court, the district court's order and memorandum addresses each of these claims in significant detail.

III. The Standard for Admitting Expert Testimony

The admissibility of expert testimony is governed by Rule 702 of the Federal Rules of Evidence:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

Fed. R. Evid. 702 (2003). Rule 702 imposes on a district court a gatekeeper obligation to "ensure that any and all scientific testimony or evidence admitted is not only relevant, but reliable." Daubert, 509 U.S. at 589. Fulfilling the gatekeeper duty requires the judge to assess the reasoning and methodology underlying the expert's opinion and determine whether it is both scientifically valid and applicable to a particular set of facts. Id. at 592-93. The Supreme Court has made clear that "where [expert] testimony's factual basis, data, principles, methods, or their application are called sufficiently into question . . . the trial judge must determine whether the testimony has 'a reliable basis in the knowledge and experience of [the relevant] discipline.'" Kumho Tire, 526 U.S. at 149 (quoting Daubert, 509 U.S. at 592).

To be reliable under Daubert, an expert's scientific testimony must be based on scientific knowledge, which "implies a grounding in the methods and

procedures of science" based on actual knowledge, not mere "subjective belief or unsupported speculation." 509 U.S. at 590. In other words, "an inference or assertion must be derived by the scientific method . . . [and] must be supported by appropriate validation--*i.e.* 'good grounds,' based on what is known." Id. While expert opinions "must be based on facts which enable [the expert] to express a reasonably accurate conclusion as opposed to conjecture or speculation, . . . absolute certainty is not required." Gomez v. Martin Marietta Corp., 50 F.3d 1511, 1519 (10th Cir. 1995) (quotation omitted). "The plaintiff need not prove that the expert is undisputably correct or that the expert's theory is 'generally accepted' in the scientific community." Mitchell v. Gencorp Inc., 165 F.3d 778, 781 (10th Cir. 1999). Instead, the plaintiff must show that the method employed by the expert in reaching the conclusion is scientifically sound and that the opinion is based on facts that satisfy Rule 702's reliability requirements. Id.

To assist in the assessment of reliability, the Supreme Court in Daubert listed four nonexclusive factors that the trial court may consider: (1) whether the opinion at issue is susceptible to testing and has been subjected to such testing; (2) whether the opinion has been subjected to peer review; (3) whether there is a known or potential rate of error associated with the methodology used and whether there are standards controlling the technique's operation; and (4) whether the theory has been accepted in the scientific community. 509 U.S. at 593-94. As

noted, the list is not exclusive, and district courts applying Daubert have broad discretion to consider a variety of other factors. Kumho Tire, 526 U.S at 150 ("[W]e can neither rule out, nor rule in, for all cases and for all time the applicability of the factors mentioned in Daubert . . . . Too much depends upon the particular circumstances of the particular case at issue.").

Generally, the district court should focus on an expert's methodology rather than the conclusions it generates. Daubert, 509 U.S. at 595. However, an expert's conclusions are not immune from scrutiny: "A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered." Joiner, 522 U.S. at 146 ("[N]othing in either Daubert or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert."). In Joiner, when faced with a claim that the experts in question relied on studies that did not support their conclusions, the Supreme Court reviewed each study and concluded that the district court did not abuse its discretion in excluding the testimony. Id. at 145-46 ("[I]t was within the District Court's discretion to conclude that the studies upon which the experts relied were not sufficient, whether individually or in combination, to support their conclusions . . . .").

Under Daubert, "any step that renders the analysis unreliable . . . renders the expert's testimony inadmissible. This is true whether the step completely

changes a reliable methodology or merely misapplies that methodology." Mitchell, 165 F.3d at 782 (quoting In re Paoli R.R. Yard PCB Litigation, 35 F.3d 717, 745 (3d Cir. 1994)). It is critical that the district court determine "whether the evidence is genuinely scientific, as distinct from being unscientific speculation offered by a genuine scientist." Id. at 783 (quoting Rosen v. Ciba-Geigy Corp., 78 F.3d 316, 318 (7th Cir. 1996)). Regardless of the specific factors at issue, the purpose of the Daubert inquiry is always "to make certain that an expert, whether basing testimony upon professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." Kumho Tire, 526 U.S. at 152.

IV. Did the Medical Literature Support the General Causation Opinion?

A. The Railroad's Argument

The Railroad first argues that the district court abused its discretion because Dr. Teitelbaum's general causation opinion was not supported by the medical literature he cited. The district court specifically found that Dr. Teitelbaum's methodology--i.e., surveying medical literature, drawing general propositions and then applying them to arrive at a conclusion of general causation--was reliable. III R. at 664. The Railroad does not contend that this methodology is per se unreliable, Aplt. Br. at 29, but instead argues that an

otherwise reliable method was applied to the facts of this case in an unreliable manner. First, the Railroad suggests that Dr. Teitelbaum erroneously reached certain "generally accepted propositions" regarding the symptoms, causes and typical occurrences of HACE only by selecting and relying on portions of the literature that were favorable to his ultimate conclusion without explaining or even considering unfavorable portions. Second, it argues that Dr. Teitelbaum then unjustifiably extrapolated from these erroneously drawn general propositions to conclude that general causation existed here. Aplt. Br. at 21, 30-38.

B. What the District Court Found

After conducting a "thorough review of the articles," the district court found that Dr. Teitelbaum's reliance on them for various general propositions was proper and that his conclusions were "adequately supported by the scientific literature." III R. at 664. The court emphasized that "[a]nalyzing each individual article and requiring that each article fully support Dr. Teitelbaum's theory, instead of focusing on the cumulative weight of the evidence, would be overemphasizing [his] conclusions, as opposed to his methodology." Id. at 665. The court found that "this is not [a] case" where "too great an analytical gap" existed between the data and the opinion. Id. After noting that the effects of each component of Mr. Goebel's injury--high altitude, oxygen deficiency and diesel fume exposure--had been individually studied and widely accepted in the

medical community and that neither the court nor the Railroad had uncovered evidence suggesting an opposite conclusion, the court concluded that Dr. Teitelbaum's opinion was reliable: "Dr. Teitelbaum's methodology is scientifically sound, and . . . his opinion reasonably flows from the data upon which he purportedly relies." Id. at 667.

C. Did the District Court Abuse its Discretion?

In arguing that the district court's review of the literature was insufficient, the Railroad directs us to specific passages in the articles that, in its view, undercut Dr. Teitelbaum's general propositions and his ultimate conclusions. Aplt. Br. at 30-38. As expected, Mr. Goebel reviews the same articles and, like the district court, reaches a conclusion in direct opposition to the Railroad. Aplee. Br. at 38-52. Notable, however, is that the Railroad apparently agrees with the district court that there is no requirement that each individual article must fully support Dr. Teitelbaum's precise theory. Aplt. Br. at 29; see also Joiner, 522 U.S. at 146-47 (noting that studies may support a conclusion either "individually *or in combination*") (emphasis added); III R. at 665-66 (collecting cases).

The Railroad's core argument is that the district court incorrectly concluded that "this is not [a] case" where "too great an analytical gap" existed between the data and the opinion. III R. at 665. When faced with such a claim, we must, as

did the Supreme Court in Joiner, review the literature to determine whether the district court was within its discretion in finding an adequate link between the existing data and the conclusions. 522 U.S. at 145-46. Given the lack of scientific literature directly addressing the confluence of all of the factors at issue in the tunnel, such a review is all the more important here.

As we stated above, our review is deferential--only if we are convinced that the district court "made a clear error of judgment or exceeded the bounds of permissible choice in the circumstances" will we disturb its ruling. Dodge, 328 F.3d at 1223 (quoting Atlantic Richfield Co. v. Farm Credit Bank of Wichita, 226 F.3d 1138, 1163-64 (10th Cir. 2000)). Despite our expertise on the law, our role as judges is not to second-guess well qualified and highly trained medical experts on difficult judgment calls within their field of expertise; our role is merely to ensure that the district court did not abuse its discretion by concluding that the expert testimony in this case was admissible under the standards outlined above. Neither the district court nor this court is in a position to declare or even to know with any degree of certainty whether otherwise admissible expert testimony is, in fact, correct. See, e.g., Daubert, 509 U.S. at 596 ("Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence."). We also approach our review mindful that the district court here had

the added benefit of the Railroad's voir dire of Dr. Teitelbaum and its cross examination of him at trial.

After a careful review of the Railroad's arguments, Dr. Teitelbaum's affidavits, the underlying medical literature and the record as a whole, we perceive no basis to conclude that the district court abused its discretion by ruling that Dr. Teitelbaum's opinion was adequately supported by the scientific literature. Given the record before us, we too are unpersuaded that this is a case where too great a gap exists between the proffered expert opinion and the underlying data.

(1) The Symptoms of HACE

The Railroad first argues that Dr. Teitelbaum's basic contention--that Mr. Goebel exhibited symptoms classically associated with HACE--is not supported by the cited literature. Aplt. Br. at 30. Among the many symptoms exhibited by Mr. Goebel were a severe headache, tightness in his chest, significant aches and soreness, shortness of breath, nausea and disorientation that rendered him unable to read. In the Railroad's view, the studies cited by Dr. Teitelbaum do not support a diagnosis of HACE based on these symptoms. After reviewing the Railroad's citations to various studies and uncovering no significant support for its position, we conclude that the district court did not abuse its discretion.

Dr. Teitelbaum's opinion--that Mr. Goebel suffered from acute mountain

sickness ("AMS") which developed into non-acute HACE, see II R. at 356, ¶ 8--was supported by The Lake Louise Consensus on the Definition of Altitude Illness, available in Thomas E. Dietz, High Altitude Medicine Guide (2000), II R. at 398. That study indicates that in the setting of a recent altitude gain, a headache plus nausea is alone sufficient to support a diagnosis of AMS. Id. Mr. Goebel clearly satisfied this criteria. The study also states that in such a setting, the presence of a "change in mental status" in a person with AMS is sufficient to support a diagnosis of HACE. Id. The included Lake Louise Consensus worksheet makes it clear that Mr. Goebel's disorientation is also sufficient to constitute a change in mental status. Id. at 400. Thus, contrary to the Railroad's suggestion, the Lake Louise Consensus supports Dr. Teitelbaum's conclusions.

In support of its argument, the Railroad also directs us to an isolated statement in Thomas E. Dietz, Altitude Illness Clinical Guide for Physicians, High Altitude Medicine Guide (2000), II R. at 403-12, where the author notes that "I have not yet seen a case of HACE in which the patient didn't ascend with AMS symptoms." Id. at 408. The Clinical Guide admittedly relies on the Lake Louise Consensus guidelines relating to symptoms and diagnosis of altitude illnesses. Id. at 403, 407. Because under those guidelines Mr. Goebel exhibited symptoms of AMS *and* HACE, the quoted statement relied upon by the Railroad is utterly irrelevant. When read as a whole, the portions of the High Altitude Medicine

Guide contained in the record support Dr. Teitelbaum's opinion. See id. at 416-18 (discussing the symptoms associated with AMS and HACE and essentially echoing the conclusions of the Lake Louise Consensus).

Another of the Railroad's contentions is that the chapter in Michael P. Ward et al., High Altitude Medicine and Physiology 412-18 (2d ed. 1995), entitled "High Altitude Cerebral Edema and Retinal Haemorrhage," II R. at 333, contradicts Dr. Teitelbaum's opinion. In fact, in discussing the typical symptoms associated with AMS and HACE, this chapter once again echoes the conclusions of the Lake Louise Consensus. Id. (symptoms of AMS include headache and nausea; clouding of consciousness indicates HACE). Contrary to the Railroad's suggestion, Aplt. Br. at 31, this chapter at no point states that "loss of consciousness" is among the "*classic* symptoms of HACE." Id. (emphasis in original). Although the chapter states that "[o]ften there is also an element of pulmonary edema" exhibited in HACE sufferers, II R. at 334, it is undisputed that Mr. Goebel did not exhibit signs of that condition. Nonetheless, this quite general and self-limited statement is insufficient to render Dr. Teitelbaum's conclusion unreliable given the obvious corollary that cases must exist where no signs of pulmonary edema are exhibited by a HACE sufferer.

A similar issue arises from the Railroad 's citation to the Statement on High Altitude Illnesses, Canada Communicable Disease Report (Nov. 15, 1998), II R.

at 424, which states that HACE "rarely occurs" without high altitude pulmonary edema. Id. at 430. Although this is a stronger statement than that made in the High Altitude Medicine and Physiology chapter just discussed, it still fails to contradict materially Dr. Teitelbaum's opinion. Once again, this statement is limited by its own terms, i.e., "rarely." Id. In addition, the Statement continues by stating that HACE is characterized by symptoms including an "altered level of consciousness in the form of confusion [and] impaired thinking." Id. at 431. Its discussion of AMS again relies on the parameters listed in the Lake Louise Consensus. Id. at 427. Therefore, when read as a whole, the Report does not cast serious doubt on Dr. Teitelbaum's opinion and is certainly insufficient as a basis for finding that the district court abused its discretion by concluding otherwise.

The Railroad's attempted reliance on isolated passages and concepts in the following sources is similarly unavailing: Peter H. Hackett, The Cerebral Etiology of High-Altitude Cerebral Edema and Acute Mountain Sickness, 10 Wilderness and Envtl. Med. 97 (1999), II R. at 446; M. Jay Porcelli & Gary M. Gugelchuk, A Trek to the Top: A Review of Acute Mountain Sickness, 95 J. Am. Osteopath Ass'n. 718 (1996), II R. at 309; and Phillip R. Yarnell et al., High-Altitude Cerebral Edema (HACE): The Denver/Front Range Experience, 20 Seminars in Neurology 209 (2000), II R. at 535. When read as a whole, none of these articles calls Dr. Teitelbaum's opinion into question. In fact, our review of them

substantially buttressed Dr. Teitelbaum's analysis given their consistency with the Lake Louise Consensus parameters. Our review of Railroad expert Dr. Neil Rosenberg's affidavit, III R. at 599-609, likewise provides no additional evidence that Dr. Teitelbaum's opinion is not supported by the cited studies; Dr. Rosenberg's affidavit falls well short of a showing that would allow us to conclude that the district court abused its discretion.

(2) Developing HACE at or near 9,200 feet

The Railroad also apparently argues that the studies cited by Dr. Teitelbaum do not support a conclusion that it is even possible for a person to develop HACE at altitudes at or near 9,200 feet. Aplt. Br. at 35-36; id. at 35 (reiterating Dr. Rosenberg's opinion that the altitudes attained by Mr. Goebel are not capable of causing HACE). In the Railroad's view, the studies demonstrate that "HACE is very rare and occurs at altitudes well above the 9,200 feet of Moffat Tunnel." Id. at 36. While subtle inconsistencies might well exist across the various studies noted by the Railroad, we are unable to conclude that the district court abused its discretion by finding that the studies as a whole supported Dr. Teitelbaum's opinion.

It is worthwhile to note first that none of the studies pointed to by the Railroad state that a person *cannot* develop HACE at altitudes of 9,200 feet. On the contrary, the studies in the record largely agree that it is possible to suffer

from altitude illnesses such as AMS and HACE at such elevations. The consensus appears to be that high-altitude illnesses are an issue to be considered for humans at altitudes of only 5,280 feet, the beginning of what is commonly referred to as "high altitude" elevation. M. Jay Porcelli & Gary M. Gugelchuk, A Trek to the Top: A Review of Acute Mountain Sickness, 95 J. Am. Osteopath Ass'n. 718, 718 (1996), II R. at 309; All About Altitude Illness, High Altitude Medicine Guide, II R. at 415 (noting that high altitude begins at or near 5,000 feet).

The studies also agree that although high altitude illnesses *can* affect people at altitudes around only 5,000 feet, such illnesses are rare below 8,000 feet. All About Altitude Illness, II R. at 415, 417; Statement on High Altitude Illnesses, Canada Communicable Disease Report, II R. at 424 (noting that "some susceptible individuals may experience symptoms of altitude-related illness beginning as low as 2,500 m[eters]."); Altitude Illness Clinical Guide for Physicians, High Altitude Medicine Guide, II R. at 405. However, the studies make it clear that above the 8,000 foot line, high altitude illnesses are a real concern and become much more likely to cause people problems. E.g. American Academy of Family Physicians, High-Altitude Illness: How to Avoid It and How to Treat It (1998), II R. at 499. Therefore, the medical literature in the record lends ample support to the conclusion that Mr. Goebel could suffer from high altitude illnesses at elevations of 9,200 feet.

What is even more critical, however, is that Dr. Teitelbaum's opinion is not that Mr. Goebel developed HACE solely from the altitude exposure; rather, he opined that other factors contributed to the onset, including the oxygen context of the air, the heavy diesel-fume pollution, the counterproductive use of the respirator, Mr. Goebel's increased activity during the incident, and Mr. Goebel's individual physiologic response. II R. at 245. No individual study in the record attempts to account for and control additional factors such as those relied upon by Dr. Teitelbaum. The Railroad's argument based on studies relying solely on altitude as the cause of a given high altitude illness therefore misses the point. See Aplt. Br. at 35 (citing N. A. Lassen, Increase of Cerebral Blood Flow at High Altitude: Its Possible Relation to AMS, 13 Int'l J. Sports Med. S47 (1992) (dealing with altitudes of 12,000 to 15,000 feet), II R. at 298). The district court did not abuse its discretion by concluding that the studies supported Dr. Teitelbaum's opinion that Mr. Goebel could have developed AMS and HACE at an elevation near 9,200 feet.

(3) Onset of HACE in One Hour

The Railroad's final argument relating to the studies is that "[n]othing in the medical or scientific literature suggests that HACE can be developed in an exposure of less than one hour." Aplt. Br. at 37. In the Railroad's view, although HACE would ordinarily require one to three days to develop, onset might possibly

occur after 12 hours but is simply not going to manifest in the span of one hour. Id. We perceive more than one error in the Railroad's argument.

First, we disagree with the Railroad's characterization of the facts. According to its own statement of facts, on the night of the incident Mr. Goebel started work at 7:30 p.m. at an altitude of approximately 5,198 feet (i.e., in Denver). Aplt. Br. at 3-4; I R. at 99. He was directed to operate his helper locomotives through the Moffat Tunnel at elevations near 9,200 feet and to wait on the other side at Tabernash, elevation 8,318 feet. Id. The Railroad states that five hours (i.e., around 12:30 am) after he started work in Denver, Mr. Goebel had traversed the tunnel and sat waiting on a siding at Winter Park, elevation approximately 9,100 feet. Aplt. Br. at 4; I R. at 99. After meeting the train he was assigned to assist and experiencing the tunnel incident, Mr. Goebel descended to Rollinsville, elevation 8,367 feet, to await an ambulance. Aplt. Br. at 8; I R. at 99. At 2:50 a.m., more than seven hours after he began the ascent in Denver, Mr. Goebel was placed on pure oxygen en route to the hospital. Aplt. Br. at 8.

Consistent with our earlier observation from the studies, Mr. Goebel was technically at "high altitude" elevations from the moment he began his ascent from Denver. Although it is not clear precisely how long Mr. Goebel spent at elevations over 8,000 feet, it is absolutely clear that he was exposed for far longer

than one hour to elevations where altitude illnesses are a real concern. The Railroad confuses two important timelines. The first is Mr. Goebel's exposure only to high altitudes in excess of 8,000 feet, which was clearly well over one hour. The second is his exposure to various added factors inside the tunnel, including the diesel-fume polluted atmosphere and his counterproductive use of an ineffective respirator. This second window of time was indeed approximately one hour.

This important distinction focuses us on the second and most critical error in the Railroad's argument. Once again, the Railroad fails to address directly the substance of Dr. Teitelbaum's opinion. Dr. Teitelbaum has never claimed that Mr. Goebel developed HACE solely from the altitude exposure; instead, Dr. Teitelbaum has repeatedly averred that the high altitude and the other factors *combined* to produce the onset of HACE. II R. at 245. The Railroad's failure to recognize and address this fact renders unpersuasive its citation to studies for the proposition that altitude exposure *alone* does not *usually* lead to HACE in under 12 hours. Aplt. Br. at 36-37. It is clear that Mr. Goebel was exposed to altitudes exceeding 8,000 feet for much longer than one hour. More importantly, he was inside the tunnel at altitudes exceeding 9,000 feet for approximately one hour and was subjected to a variety of other factors that, in Dr. Teitelbaum's view, significantly worsened his condition. The studies cited by the Railroad do not

render Dr. Teitelbaum's opinion unreliable and do not give us reason to conclude that the district court abused its discretion.

V. Was the Differential Diagnosis Reliable?

A. The Railroad's Argument

The Railroad's second major contention is that the district court abused its discretion because Dr. Teitelbaum's specific causation testimony was based upon an unreliable differential diagnosis that failed to account for obvious alternative explanations. The Railroad's theory is that Mr. Goebel's cognitive deficits were the result not of brain damage caused by HACE, but were merely symptoms of depression or emotional distress. Therefore, the symptoms Mr. Goebel suffered during and after the tunnel incident--i.e., dizziness, headache, abdominal pain, back pain, and disorientation--were not sufficient to rule in HACE as a possible diagnosis; instead, the obvious alternative was that Mr. Goebel suffered only from overexposure to diesel fumes, which in the Railroad's view is a diagnosis more in line with the symptoms, but one which it acknowledges would not account for lasting cognitive deficits from brain damage. Aplt. Br. at 41-42 (citing overexposure to diesel smoke as an example of something "not capable of causing brain damage").

Upon closer inspection, much of the Railroad's differential diagnosis argument is largely a reiteration of its first argument. Recognizing that a reliable

differential diagnosis is admissible in this circuit given a valid showing of general causation, see Hollander v. Sandoz Pharms. Corp., 289 F.3d 1193, 1210-11 (10th Cir. 2002), the Railroad spends considerable effort rehashing its view that the scientific literature does not support Dr. Teitelbaum's conclusions. See Aplt Br. at 43-44, 47, 49, 51. In the alternative, the Railroad argues that even if general causation were established, Dr. Teitelbaum's differential diagnosis was unreliable for failing to account for obvious alternative explanations, i.e., overexposure to diesel fumes and depression. Id. at 47-48.

B. What the District Court Found

Citing Hollander and its ruling in favor of Mr. Goebel on the general causation issue, the district court first dismissed the Railroad's argument that the differential diagnosis was automatically insufficient because no general causation was shown. III R. at 669. Then, noting that Dr. Teitelbaum reviewed Mr. Goebel's medical history, conducted a physical exam, reviewed outside information, considered medical studies and theories in accepted medical literature, relied on further testing performed by other specialists, and eliminated other possible diagnoses, the district court found that Dr. Teitelbaum followed "standard medical procedure in evaluating and diagnosing" Mr. Goebel and that this methodology was based on valid scientific method. Id. at 670.

Addressing the Railroad's contention that Mr. Goebel did not exhibit

certain classic symptoms of HACE, the court found that Dr. Teitelbaum nonetheless reached a reliable differential diagnosis in concluding that Mr. Goebel's symptoms correlated both with HACE and overexposure to diesel fumes. Id. at 672. The court relied on Dr. Teitelbaum's reasoning that, because Mr. Goebel suffered a less than acute form of HACE, certain symptoms associated with acute HACE would not have necessarily manifested in Mr. Goebel. Id. Having concluded that Dr. Teitelbaum reliably applied a sound methodology, the court held that Mr. Goebel's failure to exhibit certain symptoms of acute HACE was simply a matter for the trier of fact to consider when assigning weight to the testimony.

Regarding the Railroad's argument that Dr. Teitelbaum failed to rule out depression as an obvious alternative cause, the district court first noted that several circuits have held that the failure to rule out *all* possible alternative causes of an illness does not automatically render an expert's testimony inadmissible. Id. at 674. After again noting that Dr. Teitelbaum followed sound scientific method in making his diagnosis, the district court held that his failure to exclude explicitly one alternative (depression) did not affect the admissibility of the testimony, although it was an issue the fact finder could consider when assigning weight. Id. at 675. The court buttressed its conclusion by pointing to the temporal relationship between the incident and Mr. Goebel's symptoms and by

noting the fact that even the Railroad's experts could not definitively opine that Mr. Goebel's cognitive deficits were caused by depression. Id. at 676.

C. Did the District Court Abuse its Discretion?

Whatever the merits of differential diagnosis in the abstract, the district court correctly determined, based on our precedent in Hollander, that it can admit a differential diagnosis that it concludes is reliable if general causation has been established. Hollander, 289 F.3d at 1210 (declining to decide if differential diagnosis is reliable in general but stating that such a diagnosis is admissible in certain circumstances if determined to be reliable). We have already concluded that the district court did not abuse its discretion by concluding that Dr. Teitelbaum's general causation opinion was admissible. Therefore, as in Hollander, the only issue that remains is whether the district court abused its discretion by concluding that Dr. Teitelbaum's specific differential diagnosis was reliable. In Hollander, we recognized that a differential diagnosis is most useful when "the party relying on the diagnosis has offered independently reliable evidence that the allegedly dangerous drug or substance had harmful effects," i.e., when a reliable general causation opinion has been offered to rule the substance in as a potential cause. Id.

Having determined that Dr. Teitelbaum's general causation opinion was reliable and therefore admissible, we now conclude that the district court

correctly ruled that Dr. Teitelbaum's differential diagnosis was also reliable because he followed a standard and accepted methodology in arriving at the diagnosis, he adequately explained why Mr. Goebel might not exhibit every symptom of acute HACE, and he adequately considered and ruled out some alternative explanations even if he did not explicitly rule out depression.

The district court correctly relied on the temporal relationship between the tunnel incident and Mr. Goebel's symptoms as just one *factor* that supported Dr. Teitelbaum's conclusion. The court is not permitted to, and did not, rely on the temporal relationship by itself as evidence of causation. See, e.g., Heller v. Shaw Indus., Inc., 167 F.3d 146, 154 (3d. Cir. 1999) ("The temporal relationship will often be (only) one factor, and how much weight it provides for the overall determination of whether an expert has 'good grounds' for his or her conclusion will differ depending on the strength of that relationship."); Westberry v. Gislaved Gummi AB, 178 F.3d 257, 265 (4th Cir. 1999). Given Mr. Goebel's testimony about his symptoms both during and after the incident that are entirely consistent both with Dr. Teitelbaum's conclusions and with a diagnosis unrelated to depression, we agree with the district court that Dr. Teitelbaum's failure explicitly to rule out depression as one possible alternative cause is not unreasonable. Furthermore, given that the Railroad's experts could not definitely testify that depression was a valid possible alternative, Dr. Teitelbaum's failure to

specifically rule it out does little to render his diagnosis unreliable.

Finally, Dr. Teitelbaum's explanation of why Mr. Goebel might not have exhibited all the symptoms of acute HACE is reasonable. As noted above, the medical studies make it clear that Mr. Goebel exhibited symptoms sufficient to justify a HACE diagnosis. In effect, Mr. Goebel exhibited some, but not all, of the symptoms associated with HACE. He also exhibited effects of lasting cognitive deficits, a finding confirmed by outside specialists. Given this combination of symptoms, it was reasonable that Dr. Teitelbaum did not diagnose Mr. Goebel with overexposure to diesel fumes because such a diagnosis would not account for lasting cognitive deficits. For these reasons, and in light of our deferential standard of review, we conclude that the district court did not abuse its discretion by ruling that Dr. Teitelbaum's differential diagnosis was reliable and therefore admissible. As the district court correctly noted, although Dr. Teitelbaum's opinion was properly admissible, any weaknesses should have been diligently pursued and exposed on cross examination.

After a careful review the district court's decision, the Railroad's arguments, and the underlying medical literature, we cannot conclude that the district court, in admitting Dr. Teitelbaum's general causation opinion and differential diagnosis, "made a clear error of judgment or exceeded the bounds of permissible choice in the circumstances." Dodge, 328 F.3d at 1223 (quoting

Atlantic Richfield Co. v. Farm Credit Bank of Wichita, 226 F.3d 1138, 1163-64 (10th Cir. 2000)).

VI. Appellate Review of Adequate Scientific Support.

The dissent contends that this court has failed to take the correct approach by not considering whether adequate scientific support exists for Dr. Teitelbaum's opinion. The dissent concludes that the scientific support is inadequate to demonstrate that the HACE experienced by Mr. Goebel could cause permanent brain damage and that the episode in the Moffat Tunnel could have caused HACE. The trial court, having been alerted to these gaps, should have insisted that Dr. Teitelbaum explain these gaps, or excluded his testimony.

The key to this case is the deferential standard of review–under the abuse of discretion standard, the district court's decision to admit the evidence must be characterized as manifestly erroneous to warrant reversal. See Joiner, 522 U.S. at 142-43. The lack of scientific literature directly addressing the confluence of all of the factors at issue in the tunnel is hardly surprising because such tests could not be run with human subjects. Though the review was for plain error due to a belated Daubert objection, in Mascenti v. Becker, 237 F.3d 1223 (10th Cir. 2001), this court upheld the use of an expert's opinion of brain injury from diffusion hypoxia where the plaintiff had been administered multiple drugs in combination with nitrous oxide. 237 F.3d at 1231. Just as here, the defendant challenged "the

absence of professional literature to support [the expert's] opinion and asserted conflicts between portions of [the expert's] reasoning and principles which do find support in the professional literature." Id. We concluded that admission of the expert's "opinion on the specifics of the case, in the absence of published studies on this precise combination of medications and prolonged use of nitrous oxide," was not plain error. Id. at 1234.

In Mascenti, the "[d]efendant's positions disputing [the expert's] opinions were energetically developed at trial through cross-examination of [the expert] and through the testimony of defendant's own experts, *inter alia*." Id. at 1231. Contrast that with this case where the Railroad did not appeal the lack of an evidentiary hearing on remand, it never deposed Dr. Teitelbaum, and at trial it did not put on its own expert to directly refute Dr. Teitelbaum's testimony. The Railroad's theory is that exposure to diesel smoke and depression accounted for Mr. Goebel's condition, and that we as lawyers and judges on appeal can determine what the medical literature says and its import as well as any expert, let alone the trial court. Joiner did not go that far, nor did it invest this court with plenary review that would displace the discretion of the district court.

Here, adequate support for the HACE diagnosis exists, based upon the disorientation and the AMS diagnosis. Even the dissent's review of the literature contains self-limiting terms about symptoms, and quotes statements that support

Dr. Teitelbaum's conclusions. Dissent at 6-7. Though the dissent faults the supporting literature as not being verifiable, it is a little far afield for this court (not the experts) to refute the validity of the underlying data in support.

The dissent also contends that Dr. Teitelbaum did not adequately prove the onset of HACE given a one-hour exposure to the tunnel episode. Nothing in this record is categorical about the amount of time it takes for HACE to develop, particularly given that "medical science is unsure how HACE develops." Dissent at 10 n.2. Mr. Goebel spent more than one hour at elevations above 8,000 feet, and as noted above Dr. Teitelbaum is claiming that a combination of factors, some not present in the studies, resulted in the onset of HACE. True, these factors result in hypoxia, but a permissible reading of Dr. Teitelbaum's position is that the entire transition from Denver's altitude to the Moffat Tunnel apex was sufficient to cause AMS and HACE in combination with the other factors.

Conclusion

For the foregoing reasons, we conclude that the district court did not abuse its discretion by ruling that Dr. Teitelbaum's testimony was admissible under the standards of Rule 702 and Daubert. Accordingly, we AFFIRM the district court's judgment.

No. 02-1391 - Goebel v. The Denver and Rio Grande Western Railroad Company
HARTZ, Circuit Judge, dissenting:

I respectfully dissent. Dr. Teitelbaum's theory of the causation of Plaintiff's injury lacks the scientific support necessary for it to be admissible at trial.

My conclusion is not a reflection on Dr. Teitelbaum's expertise as a toxicologist. He has an impressive resume. But science is no respecter of resumes. An author's resume may cause the scientist-reader to pay attention to the author's theory; but it does quite little to cause the scientist to agree with the theory. Agreement depends upon trustworthy supporting data. Dr. Teitelbaum's resume has not prevented his opinions from being properly excluded in other litigation. *See General Electric Co. v. Joiner*, 522 U.S. 136 (1997) (affirming the district court's refusal to admit into evidence Dr. Teitelbaum's opinion regarding the cause of Mr. Joiner's lung cancer).

Dr. Teitelbaum's theory is that Plaintiff has suffered "mild diffuse [permanent brain] damage," Aplt.'s App. at 356 ¶ 8, caused by high altitude cerebral edema (HACE) "of a mild nature." *Id*. Dr. Teitelbaum does not purport to be an expert on HACE itself. He has conducted no personal research on HACE, and apparently had not even had prior patients with the condition. Moreover, he admits that if Plaintiff had HACE, his case was quite unusual, perhaps unique. One of his affidavits states: "I must point out that there will

never be any epidemiological study which duplicates this extraordinary event which led to [Plaintiff's] injury. If we write a case report of this event it likely will be the only one in the literature . . . ." *Id*. at 345 ¶ 5. To justify his conclusions, Dr. Teitelbaum asserts that he is relying on well-established general principles and refers to numerous articles in the medical literature.

The majority opinion fails to take the correct approach. It makes good arguments that the medical literature cited by Defendant does not prove Dr. Teitelbaum's opinion to be wrong. The test is not, however, whether Defendant can prove Dr. Teitelbaum's opinion is wrong. The test is whether there is adequate scientific support for Dr. Teitelbaum's opinion. Dr. Teitelbaum's opinion may in fact be correct. It may be an inspired insight. But a courthouse is not the proper forum to present inspiration. Only when the insight is properly supported by research is it admissible at trial. *See Rosen v. Ciba Geigy Corp.*, 78 F.3d 316, 319 (7th Cir. 1996) ("[T]he courtroom is not the place for scientific guesswork, even of the inspired sort. Law lags science; it does not lead it.")

Determining whether an expert's opinion rests on adequate scientific support can be a daunting task for a judge. Judges are not, and should not be, scientists. But neither must judges be passive aiders and abettors of hired guns who can pronounce their "wisdom" with such self-appointed authority that a

layperson has little chance of resolving the muddle. The solution, albeit just a partial one, is to rely on opposing experts to point to the gaps in each other's chain of reasoning. As the majority opinion properly states, "[T]he responsibility ultimately falls on th[e] challenging party to inform (via the record) those of us who are not experts on the subject with an understanding of precisely how and why the expert's conclusions fail to follow from the data set." Op. at 5. But when the challenging party satisfies this requirement and points out that the data set does not support the expert's conclusion, the expert must then present a reasonable argument to the contrary. Otherwise, the court has no choice but to "conclude that there is simply too great an analytical gap between the data and the opinion proffered." *Joiner*, 522 U.S. at 146. The majority of this panel believes that Defendant failed to satisfy its obligation to point out how the data fail to support Dr. Teitelbaum's conclusion. My view, however, is that Defendant satisfied this obligation and Dr. Teitelbaum failed to respond adequately.

There are at least two major gaps in Dr. Teitelbaum's analysis. As pointed out by Defendant's experts, Dr. Teitelbaum has not produced adequate scientific support for the proposition that HACE of the nature allegedly experienced by Plaintiff can cause permanent brain damage of the type allegedly suffered by Plaintiff. And he has not produced adequate scientific support for the proposition that the episode in Moffat Tunnel could have caused HACE.

First, assuming that Plaintiff indeed suffered HACE, could his HACE have caused the brain damage allegedly present? Dr. Teitelbaum asserts that Plaintiff has mild diffuse permanent brain damage. But Dr. Neal L. Rosenberg, Defendant's expert, stated in his affidavit that if Plaintiff "had suffered a permanent injury to the brain from [the tunnel] event, he would almost certainly have been rendered unconscious (or resulting in a coma), immediately at the time of the alleged exposure." Aplt.'s App. at 581. Of course, the district court was not bound to reject Dr. Teitelbaum's opinion just because another expert disagreed. Nevertheless, once the trial court was alerted to the potential gap in Dr. Teitelbaum's chain of reasoning, it should not have admitted the expert's opinion into evidence unless the record before the court—other than a bald *ipse dixit* from the expert, *see Joiner*, 522 U.S. at 146—filled that gap.

Dr. Teitelbaum's testimony and affidavits contain no support for the proposition that Plaintiff could have suffered permanent brain injury from HACE without having lost consciousness during the episode. Indeed, Dr. Teitelbaum fails to address the issue. The closest he comes is in the following passage from one of his affidavits:

> In Ward's textbook, High Altitude Medicine and Physiology, Chapter 19 deals with high altitude cerebral edema. Defendants have confused increased intracranial pressure which results in local diffuse damage with malignant high altitude cerebral edema which causes ataxia, irrationality, hallucinations, clouding of consciousness and death. The syndrome suffered by Mr. Goebel was more severe than

> that suffered by the usual skier or tourist who reaches altitude, and less severe than that suffered by mountain climbers who have acute major irreversible damage. He suffered mild diffuse damage which in some individuals would be of little consequence and might not even be detected.
>
> However, in a railroad engineer who is required to carry out complex mechanical and intellectual tasks, these residua are disabling. The failure of the defendants to review Ward's textbook to which I referred in my affidavit and which deals with the fundamental physiology of oxygen and hemoglobin at high altitude has contributed substantially to the confusion set forth in their motion for new trial and in the course of their cross-examination of me.

Aplt.'s App. at 356. Chapter 19 of Ward's text, however, says nothing about permanent brain damage, whether mild diffuse damage or otherwise. *See* Michael P. Ward, et al., High Altitude Medicine & Physiology, 412-17 (2d ed. 1995) (Ward). The three "[t]ypical cases" it describes resulted in either death or complete recovery. *Id.* at 413-14.

In an abundance of caution, I have reviewed the literature cited by Dr. Teitelbaum (as best I can as a layman, without the assistance of any references by Dr. Teitelbaum to specific pages or passages in the literature) to see whether it fills the gap. It does not. The literature reports that death is a common consequence of untreated HACE. Victims who survive, however, generally exhibit no signs of permanent injury. The case reports in Dr. Teitelbaum's cited literature typically refer to full recovery of the victim. *See* Thomas E. Dietz, *All About Altitude Illness,* in High Altitude Medicine Guide 4, *at* http://www.high-altitude-

medicine.com/AMS.html (Dietz I) ("People with HACE . . . usually recover completely"); Peter H. Hackett, et al., *High-altitude Cerebral Edema Evaluated with Magnetic Resonance Imaging*, 280 JAMA 1920, 1920 (1998) (Hackett I) ("all [nine] patients [with HACE] completely recovered"); Mark D. Harris, et al., *High-Altitude Medicine*, 57 Am. Fam. Physician 1907, 1911 (1998) (Harris) ("usually recover completely"); Phillip R. Yarnell, et al., *High-Altitude Cerebral Edema (HACE): The Denver/Front Range Experience*, 20 Seminars in Neurology 209, 216 (2000) (Yarnell) (HACE "is completely reversible with expeditious treatment").

To be sure, one article cited by Dr. Teitelbaum says that persistence of "neurologic deficits," while "extremely rare," has been reported, Thomas E. Dietz, *Altitude Illness Clinical Guide for Physicians*, in High Altitude Medicine Guide 7, at http://www.high-altitude-medicine.com/AMS-medical.html (Dietz II); and another article, contrary to every other statement on the subject in the cited articles, states that HACE "*often* resolves with longlasting neurologic and psychiatric deficits," Michael Weidman & Geoffrey C. Tabin, *High-Altitude Retinopathy and Altitude Sickness*, 106 Ophthalmology 1924, 1926 (1999) (emphasis added). But neither of these two assertions (which, by the way, were not specifically referred to in Dr. Teitelbaum's testimony or affidavits) is accompanied by any reference to authority, such as a journal article, for its truth.

As a result, it is impossible to verify the assertions. More importantly, even assuming the truth of the assertions, the absence of supporting references makes it impossible to compare those victims' experiences with HACE to Plaintiff's experience, to see whether, for example, the victims suffered permanent brain damage without having been rendered unconscious by HACE.

In sum, Dr. Teitelbaum's literature provides no support for any person's having suffered permanent brain injury—much less "mild diffuse damage"—from a case of HACE that had not resulted in symptoms (such as coma) significantly more severe than those experienced by Plaintiff. We have no basis for assuming that the reports of permanent neurological damage referred to in Dr. Teitelbaum's literature were related to cases of "mild HACE" (as Dr. Teitelbaum describes what happened to Plaintiff). On this ground alone, Dr. Teitelbaum's opinion of the cause of Plaintiff's alleged brain injury has no business in a court of law.

The second gap in Dr. Teitelbaum's opinion relates to the cause of HACE. Dr. Teitelbaum asserts that the tunnel episode, which lasted less than 60 minutes, caused Plaintiff to suffer HACE. Dr. Rosenberg, however, said in his affidavit: "HACE typically requires 1 to 3 days to develop . . ., and I am unaware of a single case of HACE developed in less than 12 hours." Aplt.'s App. at 608. Again, Dr. Teitelbaum does not confront this challenge. The closest he comes on this issue is the following passage in an affidavit:

> Mr. Goebel's transition in a short period of time from Denver's altitude of approximately 5,280 feet to the Moffat Tunnel apex altitude of 9,200 feet is more than sufficient to produce both acute mountain sickness (AMS) and high altitude cerebral edema (HACE) in an individual who is both sensitive to the development of the disease and is exposed to factors in addition to the altitude stresses which contribute to the problem. I have discussed all of these stressors and toxicity issues in my prior declarations.

Aplt.'s App. at 251. He cites no literature to support the proposition that HACE could be caused during the brief period in the tunnel.

Again, in an abundance of caution I read the various articles attached to his affidavits. None of the articles comes close to suggesting that HACE can arise during a 60-minute episode of high-altitude hypoxia. In one article cited by Dr. Teitelbaum all nine reported cases of HACE occurred after the victim had spent two or more days at high altitude. Hackett I, *supra,* at 1922. In another, all 13 reported cases involved patients who had spent at least three days at high altitude. Yarnell, *supra*, at 212. Other articles cited by Dr. Teitelbaum contain the following statements:[1]

> 1. "The 6-to-96-hour delay between the arrival at a high altitude and the onset of symptoms . . . ." George H. Sands, et al., *Cough, Exertional & Other Miscellaneous Headaches*, 75 Med.

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[1]The articles often speak of the more general ailment AMS—acute mountain sickness—which can progress to the more severe ailment HACE. *See* Ward, *supra*, at 412 ("The malignant form of AMS we call . . . HACE"); *The Lake Louise Consensus on the Definition of Altitude Illness*, in High Altitude Medicine Guide, at http://www.high-altitude-medicine.com/AMS-LakeLouise.html ("HACE [c]an be considered 'end stage' or severe AMS"); Dietz I, *supra*, at 4 (HACE is at the "severely ill" end of AMS spectrum).

Clinics 733, 742 (1991) (Sands).

2. "Progression to HACE from mild AMS varies from 12 hours to the more common duration of between 1 and 3 days." Committee to Advise on Tropical Medicine and Travel, *Statement on High-Altitude Illnesses,* 24 Can. Communicable Disease Rep. 1, 8 (1998) (Canada Statement).

3. "AMS affects climbers 6 h[ours] to several days after an ascent to an altitude greater than 3,000 m [9800 feet]." Sarper Karaküçük & G. Erutgral Mizra, *Ophthalmological Effects of High Altitude,* 2000 Ophthalmic Res. 30, 31(1999).

4. HACE is assumed to occur with "severe prolonged hypoxia." John W. Severinghaus, *Hypothetical roles of angiogenesis, osmotic swelling, and ischemia in high-altitude cerebral edema,* 79 J. Applied Physiology 375, 375 (1995) (Severinghaus I).

5. "Is the increase of CBF [cerebral blood flow] [from acute hypoxia] a causative factor in acute mountain sickness[?] This is highly unlikely. The increase in blood flow is almost instantaneous, while AMS, more specifically the headache, sets on only after hours." N. A. Lassen, *Increase of Cerebral Blood Flow at High Altitude: Its Possible Relation to AMS*, 13 Int. J. Sports Med. 47, 48 (1992).

6. "[V]asogenic edema develops in humans (and sheep) who become moderately ill with AMS/HACE during 24 hr or more of hypoxic exposure." Peter H. Hackett, *The cerebral etiology of high-altitude cerebral edema and acute mountain sickness*, 10 Wilderness & Envtl. Med. 97, 106 (1999).

Dr. Teitelbaum may have been assuming that the severity of the hypoxia suffered by Plaintiff accounts for the quick onset. But the literature does not support such an assumption. According to the literature cited by Dr. Teitelbaum, HACE appears to be an *indirect* effect of prolonged hypoxia, which can be distinguished from direct effects of oxygen deprivation on the brain. *See, e.g.,*

Canada Statement, *supra*, at 2-3 (describing acute hypoxia and AMS as distinct ailments). Unlike what happens in AMS or HACE, "Acute severe hypoxia may, within minutes, induce headaches, nausea, and vomiting." Severinghaus I, *supra*, at 377. As explained in another article, "AMS is now considered to be primarily due to the body's response to modest hypoxia and has a different pathophysiology from simple acute hypoxia, being associated with fluid shifts not seen with hypoxia alone." Canada Statement, *supra*, at 3. In noting the difference between HACE and the direct effects of oxygen deprivation, a third article said that "attempts to correlate AMS/HACE . . . with the degree of hypoxemia[] has been relatively fruitless . . . ." Peter H. Hackett, Hypoxia: Into the Next Millennium 25 (1999). And a fourth article concluded that "[t]he 6-to 96-hour delay between the arrival at a high altitude and the onset of symptoms suggests that hypoxia is not the immediate cause of AMS." Sands, *supra*, at 742.[2]

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[2]Medical science is unsure how HACE develops, *see* John W. Severinghaus, *Uses of High Altitude for Studies of Effects of Hypoxia,* in Oxygen Transport to Tissue XX 17, 24 (1998) ("[t]he underlying pathophysiology of HACE . . . is still poorly understood"). (This in itself renders unacceptably speculative Dr. Teitelbaum's theory that HACE could be caused by a combination of factors never before reported or, as far as one can tell, observed to accompany HACE.) Nevertheless, a brief description of one of the theories may help to understand how HACE can differ from the direct effects of hypoxia. One expert suggests that HACE is caused by angiogenesis—"[t]he process by which growing ischemic or hypoxic tissues stimulate the in-growth of capillaries." Severinghaus I, *supra*, at 377. This expert theorizes that HACE is observed upon the "onset of angiogenesis after hours or days [of hypoxia]." *Id*. at 378. "Angiogenic capillary breakdown and the resulting edema," he says, "presumably occur only after many hours of sustained hypoxia." *Id*. at 377; *see* Yarnell, *supra*, at 216

Despite Dr. Teitelbaum's failure to explain the uniquely rapid onset of Plaintiff's alleged HACE, the majority opinion makes an attempt. It asserts that we should look not just at the time in the tunnel but also should consider the entire train trip from Denver, which began at the high altitude of 5,280 feet. Op. at 20-22. This assertion, however, ignores the medical literature. The articles say, for example, that "[p]ractically speaking . . . , we generally don't worry about elevations below about 2500 m (8000 ft) since altitude sickness [much less HACE] rarely occurs lower than this." Dietz I, *supra*, at 1. When one article says that "some susceptible individuals may experience symptoms of altitude-related illness beginning *as low as* 2500 m [8200 feet]," Canada Statement, *supra*, at 1 (emphasis added), I infer that conditions below 8200 feet simply do not induce AMS (much less HACE) in anyone. *See also* Yarnell, *supra*, at 211 ("In general, . . . high-altitude illness [is] associated with rapid ascent above 8202 feet (2500 m)."). According to the literature, the mean altitude at which HACE develops is 15,500 feet in cases where, as here, the person does not also suffer high altitude pulmonary edema (HAPE). (For those who suffer HAPE, the mean altitude is 12,850 feet. *Id*. at 214.)

Furthermore, the cited articles report that acclimation to high altitude is the key to avoiding AMS and HACE. *See* Dietz II, *supra*, at 3 ("continued ascent is acceptable" once one is free of AMS symptoms); Harris, *supra*, at 1907 (to avoid

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("Angiogenesis is stimulated within *hours* of hypoxia." (emphasis added.))

AMS, ascend only 1,000 feet per day after reaching 8,000 feet); Dietz I, *supra*, at 3 (above 10,000 feet one's sleeping elevation should not increase more than 1,000 feet per night); Canada Statement, *supra*, at 6 (safest method to prevent AMS is "graded ascent"). And the higher one's home altitude, the better one can adjust. *See* Yarnell, *supra*, at 211 (odds of getting AMS are 3.5 times greater if usual residence is below 3,000 feet). Here, the first four hours of the trip were below 8,300 feet elevation and unlikely to create the conditions for HACE, or even AMS. Also, Plaintiff would be less likely than most people to develop AMS at such an altitude because he was acclimated to the mile-high elevation of Denver, where he lived. Nor did Plaintiff experience any conditions before entering the tunnel that Dr. Teitelbaum said would exacerbate the hypoxia—exposure to thick diesel smoke, physical exertion, or use of a counterproductive respirator. In short, the majority opinion expands the time during which Plaintiff was exposed to high-altitude hypoxia by speculation not supported by, indeed contradicted by, the medical literature.

More importantly, the majority opinion's speculation is not even supported by Dr. Teitelbaum's own assertions. None of his opinions regarding the cause of Plaintiff's alleged HACE relies on Plaintiff's train trip up to the tunnel. Instead he focuses on the events in the tunnel. For example, his final affidavit states:

> 2. . . . The[] elements of causation are the altitude *of the tunnel*, the oxygen content of air at that altitude, the physiologic principles

> which govern the partial pressure of oxygen in the blood at that altitude, the presence of diesel fuel in [Plaintiff's] environment, the use of an inappropriate respirator, and the duration of the event.
>
> 3. In my opinion, the injury to [Plaintiff] was caused by an increase in intracranial pressure due to cerebral edema and a decreased oxygen supply to the brain which in turn were the result of the altitude *of the tunnel*, the oxygen content of the air, and the partial pressure of oxygen in the blood, combined with the exercise involved in his escape, the polluted atmosphere, and individual physiologic response of the patient."

Aplt.'s App. at 344 (emphasis added). Dr. Teitelbaum makes no reference to the altitudes encountered by Plaintiff on the trip *to* the tunnel. I do not think we can rescue his opinion from the HACE-causation gap by relying on a highly suspect theory that not even he has embraced.

As Defendant put the matter in its opening brief: "Selecting only favorable factoids out of articles, without considering their overall conclusions, and without confronting or explaining why the unfavorable parts of the article have been rejected, is *not* science—it is simply advocacy. And that is exactly what Dr. Teitelbaum did." Aplt.'s Br. at 29. I am afraid that description is accurate. I would reverse the district court's decision to admit Dr. Teitelbaum's testimony and order entry of judgment in favor of Defendant.